ATELECTASIS OF THE NEWBORN*

TREATMENT BY BRONCHOSCOPIC DRAINAGE

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ATELECTASIS of the newborn has always been a serious condition and one which has been very difficult to treat. Unfortunately, the usual conservative measures in treatment are often inadequate, and the infant frequently expires of exhaustion.

Your speaker wishes to present the procedure of bronchoscopic aspiration to this group as an adjunct in the treatment of severe cases of congenital atelectasis due to obstruction of the bronchi by body secretions.

In order to present a better series of cases, he will include those bronchoscoped by Dr. Howard House. We have each done about an equal number of cases and arrived at the same statistical results while working independently. Twenty-three cases have been bronchoscoped during the past eighteen months and the results have been gratifying.

Woodward and Wadden[1] presented five cases of newborn atelectasis and quoted seventeen other cases of Dr. Maurice G. Buckles that were treated by bronchoscopic aspiration. Their article stimulated the interest of some of the pediatricians in the Los Angeles area, so that now bronchoscopy has been performed on twenty-three patients who had not responded to conservative therapy, and in which exhaustion of the infant seemed inevitable. It is the belief of your speaker that all the men who have had cases will agree that the type of patient that has been helped had been adjudged almost hopeless by them.

PORTABLE INSTRUMENT

Very little equipment is necessary for the procedure and I have a portable set of instruments so the bronchoscopy can be done without moving the patient. This is very important in view of the extreme exhaustive state found in most of these infants.

PHYSIOLOGY AND PATHOLOGY

According to Brennamen,[2] in intrauterine life the child's lungs are atelectatic. Twenty minutes after birth there is a 17 cc. air capacity, and three to six hours after birth the capacity is about 36 cc. Normally it is several days before the lungs have completely expanded. This expansion becomes complete first in the anterior borders and apices. The paravertebral, central and posterior portions are the slowest to expand.

Patterson and Farr[3] present strong evidence in support of the hypothesis that the human fetal respiratory tract is not inert in utero, but is subject to rhythmic respiratory movements, during which there is a tidal flow of amniotic fluid through the bronchial tree and alveoli. Examination of lung secretions of neonatal deaths, some stillborn, showed amniotic fluid present. That this is not entirely due to passage through the birth canal, as is generally thought, is shown by the fact that some of these cases were delivered by Caesarean section. Snyder and Rosenfeld[4] showed that India ink injected into the amniotic sac is followed shortly by the appearance of this material in the pulmonary alveoli of the animal fetus.

Wilson and Farber[5] feel lack of expansion in prematures is not a failure of respiratory effort in many instances, though this may be weak, but that it is due to cohesion of moist surfaces of the air passages. This condition may, of course, be emphasized by any disturbance in the respiratory center, imperfectly developed thoracic mechanism or through obstruction of bronchi by aspiration of amniotic fluid, mucus or blood.

INDICATIONS FOR BRONCHOSCOPIC INTERVENTION

The indication for bronchoscopic itnervention is any condition in which there is mechanical obstruction of the trachea or bronchus, not correcting itself spontaneously or with the use of approved conservative therapy, and which appears to be leading to the infant's exhaustion.

SIGNS AND SYMPTOMS

The classical picture of newborn atelectasis secondary to bronchial obstruction is herein presented:

1. Progressive dyspnea with cyanosis, most marked after crying or other effort. This may often be temporarily relieved with oxygen. When these symptoms are not present, listlessless and pallor are usually noticed.
2. Suprasternal retraction with diaphragmatic tug on the lower ribs and diminished thoracic expansion on one or both sides, associated with suppressed breath sounds with or without percussion dullness.
3. Coarse inspiratory rales and areas of localized emphysema. *Coarse moist rales are an important finding and are usually absent in cases not* due to obstruction from body secretions.
4. Dehydration.
5. X-ray of the chest will usually show a rather complete atelectasis of one or more lobes. An x-ray should be made when possible in every case, but the diagnosis is made primarily on the clinical picture.

COMMENT

A case which fulfills the above requirements and which does not respond to the usual conservative therapy should be considered a candidate for bronchoscopic treatment. Because of the great variations in the clinical picture, and rapid changes in the condition of such infants, final decision as to need for treatment should rest with the bronchoscopist and pediatrician.

Twenty-three patients have been bronchoscoped with eight deaths, representing a mortaltiy of thirty-four and seven-tenths per cent (34.7 per cent). In these eight patients with unfavorable ending, the diagnosis of true congenital atelectasis due to bronchial obstruction was not established with certainty prior to bronchoscopy. With some hesitancy, however, the patients were bronchoscoped, only to reveal little bronchial secretion. Subsequent postmortem examination revealed that only two of the eight deaths were due to true atelectasis, and the remaining six patients were found to present cerebral hemorrhage, congenital aplasia of the brain, diaphragmatic hernia, pneumonia, congenital heart and massive patchy atelectasis not due to bronchial obstruction. The corrected mortality rate, after the above complications are eliminated, would be two deaths in seventeen cases of true atelectasis, or eleven and seven-tenths per cent (11.7 per cent).

Your speaker would like to emphasize that you not become overzealous in choosing cases. In view of the experience in the above instances, I feel it is imperative, prior to bronchoscopy, to rule out by every means at our disposal the various pathological entities that may resemble congenital atelectasis due to bronchial obstruction. These include asphyxia, congenital disease of the heart and blood vessels, congenital cystic lung, cerebral trauma, blood dyscrasia, pulmonary infection, diaphragmatic hernia and congenital fetal atelectasis.

Careful examination of the respiratory rate and rhythm of the infant will aid in eliminating distress due to cerebral injury.

Just what is the mechanism that makes the broncho-

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scopy successful, I am not sure. In all patients mucus has been sucked from the bronchial tree, but in only one patient have I obtained a thick, stringy mucus plug from one bronchus. If cohesion of surfaces tends to promote atelectasis, perhaps instrumentation helps to relieve this condition.

Following bronchoscopic aspiration, there is usually a progressive improvement in the infant's respiration. However, complete relief most frequently occurs six to eight hours after instrumentation. This is apparently due to removal of secretions from the larger bronchi allowing the smaller terminal branches to drain.

Newborn atelectasis most commonly occurs in premature patients. In our series, nineteen of the twenty-three patients bronchoscoped were in this category, representing eighty-two and six-tenths per cent (82.6 per cent). The smallest infant treated was a four pound, four ounce (4 lb., 4 oz.) twin. Another weighing four pounds, seven ounces (4 lb., 7 oz.) and seven weeks premature, was likewise bronchoscoped and in both instances the infants went on to normal development.

The procedure of bronchoscopy seems to produce little, if any shock to these babies. Likewise, laryngeal edema does not develop secondary to instrumentation. This is due to two factors: first, the length of time consumed in the procedure is less than four minutes, and second, the type of bronchoscope used produces little trauma to the glottic chink

The bronchoscope used is the new improved three millimeter bronchoscope devised by Dr. Simon Jesberg.[6] While offering the same inside diameter, it is about one millimeter smaller in its outside diameter than the old Jackson instrument. Consequently this instrument passes through the smallest glottic chink without difficulty, and subsequent laryngeal edema is prevented.

SUMMARY

1. The etiology of the newborn atelectasis is not fully understood.

2. Selected cases of newborn atelectasis secondary to bronchial obstruction, which fail to respond to conservative treatment, are materially benefited by bronchoscopic aspiration.

3. Most cases of atelectasis occur in premature infants.

4. Bronchoscopic aspiration of the newborn is a relatively benign procedure when properly performed.

5. The improved three millimeter Jesberg bronchoscope is the instrument of choice in cases of newborn atelectasis.

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Girolamo Fracastoro (1484-1553).—Fracastorius, as he was generally known, made his bid for fame in such varied fields as geology, astronomy, and poetry. In medicine, he is chiefly remembered by a kind of medical poem on syphilis, "Syphilidis sive de Morbo Gallico," from which the disease obtained its present name. In his book, "De contagione," Fracastorius gave expression to his idea of the germ theory in infection (seminaria contagionum), which bears a superficial resemblance to modern doctrine.—Warner's *Calendar of Medical History.*

CLINICAL NOTES AND CASE REPORTS

STAPHYLOCOCCUS AUREUS ENDOCARDITIS*

TREATMENT WITH PENICILLIN—REPORT OF CASE

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SINCE the successful employment of penicillin in the treatment of bacterial endocarditis caused by *Streptococcus viridans,* numerous reports have appeared in which the drug has been used in *Staphylococcus aureus* endocarditis, with uniformly negative results. In the case to be presented, the diagnosis of stapylococcus endocarditis is supported by the prolonged bacteremia, the character of the murmurs, and the appearance of the emboliform lesions during the course of the infection.

REPORT OF CASE

Upon admission the patient, a 23-year-old white male, gave a history of an illness, several years previous, which had been diagnosed as arthritis with fever. Palpitations were stated to occur upon exertion. The immediate complaint was pain under the left scapula and moderate fever of one day's duration. Examination revealed a soft diastolic murmur over the aortic area, a loud P_2 sound, and acute pharyngitis. X-ray examination showed a moderately enlarged left ventricle. The temperature declined from a high of 100.8° F. on admission to 98° F. on the fifth day after admission. During this period the patient received sulfadiazine and sulfamerazine. On the fifth day after admission the temperature rose abruptly to 104° F. A blood culture taken at this time showed the presence of a coagulase-positive *Staphylococcus aureus.* A diagnosis of staphylococcus endocarditis was made, and sulfadiazine and sulfamerazine were given alternately, from 1 to 2 grams every four hours, from the sixth to the eighth day. On the ninth day after admission intramuscular penicillin therapy was begun, 10,000 Oxford Units being administered every 2 hours. Treatment was continued for 14 days, with the interruption of therapy on 2 days because of the lack of penicillin. Seven blood cultures were positive up to the eighth day of penicillin therapy; thereafter 12 cultures were negative and none positive. The patient's temperature fluctuated between 98.6° F. and 100° C. during the entire period of penicillin therapy. When 1,680,000 units of penicillin had been given, the lack of further supplies forced discontinuation of its use on the 23rd day of hospitalization. At that time sulfamerazine administration was resumed for 10 days (1 gram every 6 hours), during which time the patient's temperature became normal. He was discharged on the 34th day of hospitalization.

On the tenth day small red papilliform areas were observed on both hands, one foot, the forehead and on the back. These gradually disappeared during penicillin therapy. The blood picture was within the normal range during the entire period of illness except for a moderate leukocytosis (9,900 w.b.c. per cu. mm.) at the time of the highest fever on the fifth day.

At the present time, 18 months after admission to the hospital, the patient is free from all symptoms related to this episode. Blood cultures taken during the intervening period have been uniformly negative.

SUMMARY

In this case the clinical and laboratory findings indicate a *Staphylococcus aureus* endocarditis, possibly based upon an old rheumatic lesion, which failed to respond to sulfamerazine treatment. Prolonged penicillin therapy (1,680,000 Oxford Units were given over a period of 14 days) followed by sulfamerazine for 10 days, resulted

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